CASE REPORT JSLS

Laparoscopic Cholecystectomy Associated Lethal Hemorrhage

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ABSTRACT

Background and Objectives: Blood oozing after cholecystectomy is a rare but potentially life-threatening complication. Recently, 2 patients died from this cause. The deaths resulted in criminal proceedings and medical experts were called in. The objective of this report is to describe their findings and to elucidate preoperatively unknown risk factors of bleeding.

Methods: Medical records, autopsy, and histological examination of the liver, heart, pancreas, spleen, and kidney pertaining to 2 recent cases of laparoscopic cholecystectomy were examined. Current literature on this topic was reviewed.

Results: Preoperative risks included renal insufficiency, diabetes mellitus, and cardiopathy. The histological examination, in particular of the gallbladder bed of the liver, disclosed siderosis, inflammation, and fatty degeneration. These factors supported and perpetuated blood oozing. Postoperative ultrasonography and a hemogram might have detected and prevented death.

Conclusion: Inflammatory alterations, siderosis, and fatty degeneration of the liver are risk factors of postoperative hemorrhage. Autopsy and histological examination can detect those factors. Adequate postsurgical observation is mandatory, especially for patients at risk.

Key Words: Blood oozing, Lethal hemorrhage, Histology of the liver, Laparoscopic cholecystectomy.

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INTRODUCTION

The first report on laparoscopic cholecystectomy was published in 1985.[1] Nowadays, it is the procedure of choice for removing the gallbladder.[2–4] The most frequent complications are bile-duct trauma and bleeding.[3,5] Including procedures for acute cholecystitis, the postoperative complication rate was reported to be as high as 9% to 16%.[6,7] The rate of postoperative hemorrhage is below 1%.[4] In particular, patients with liver cirrhosis are at risk.[3,8] Most frequently, the cystic artery, epigastric vessels, and the gallbladder bed were vulnerated.[9] Death after laparoscopic cholecystectomy is very rare. The rate of mortality is below 0.1% and equals the rate of conventional cholecystectomy.[9] However, few data exist on the incidence of and reasons for bleeding from the gallbladder bed of the liver. No published reports exist on life-threatening or even lethal blood oozing.[3] Therefore, we give an account of 2 patients who experienced a laparoscopic cholecystectomy and died from a seeping hemorrhage. We also disclose risk factors for continuous postoperative bleeding discerned by autopsy and histological examinations.

CASE REPORTS

Patient 1

At age 46, the patient suffered from renal insufficiency requiring dialysis, diabetes type I, hypertension, and cholecystolithiasis. Laparoscopic cholecystectomy was performed in an external hospital. It was a usual procedure that took 42 minutes. Mobilization of the gallbladder from the gallbladder bed of the liver was unproblematic, and an argon laser was used for coagulation. Postoperatively, the patient became hypotonic. Six hours after the operation, the patient's blood pressure reached 80/50 mm Hg, the patient produced cold sweat, and the wound dressing was blood-soaked. The patient was transferred to the intensive care unit, and postoperative circulatory insufficiency was the tentative diagnosis. Nine hours postoperative, the circulation stopped. Resuscitation was ineffective, and cardiogenic shock was the most favored diagnosis. Hemoglobin concentration and hematocrit were not checked postoperatively.

Patient 2

A man age 65 had undergone aortic valve replacement and suffered from diabetes mellitus type II and hypertension. Admission was due to cholecysto- and choledocholithiasis. The concrements were confirmed by endoscopic retrograde cholangiography. A papillotomy was performed and the bile duct concrements extracted. On the next day, laparoscopic cholecystectomy was performed externally. The gallbladder was inflamed, and adhesions hampered the identification of the cystic duct and cystic artery. Mobilization of the gallbladder was difficult, time consuming, and bloody. Hemostasis by electrocoagulation in the gallbladder bed of the liver was also difficult, and styptic stripes were put on the liver surface. The operation took 105 minutes. In the 16-hour period after the operation, the patient's blood pressure remained in the normal range, but the pulse rate increased to 120 beats/minute. Thereafter, the documentation of cardiac monitoring was incomplete. Nineteen hours after the operation, the patient had to be resuscitated, and he died an hour later amid the clinical picture of myocardial infarction and heart failure.

Histology

Paraffin-embedded specimens of liver, spleen, pancreas, kidney, and heart were cut into 4-μm to 5-μm sections and stained with hematoxylin-eosin (HE), Masson Goldner, Elastica van Gieson, phosphotungstic acid hematoxylin (PTAH), and Fat Red 7B.

RESULTS

Autopsy

Patient 1

The free abdominal cavity contained 2.6 liters of blood. The gallbladder was removed and coagulated blood was adherent to the gallbladder bed. The closure of the cystic artery as well as the cystic bile duct was correct. The liver tissue adjacent to the gallbladder bed had turned inhomogeneously pale. The preparation revealed no obvious bleeding source. Further findings were sparse postmortem lividity, subendocardial bleeding of the left ventricle, cardiac hypertrophy (530 grams), anemic inner organs, atrophic kidney and pancreas, and medium grade arteriosclerosis.

Patient 2

The gallbladder was removed. The abdominal cavity contained 1.1 liters of predominantly coagulated blood, especially below the liver. The cystic duct and the cystic artery were regularly closed by clips. Blood was adherent to the liver surface. Several clips were found in the gallbladder bed. Further findings were minor bleeding into the liver tissue adjacent to the gallbladder bed, local necrosis, and fatty liver degeneration **(Figure 1)**. In addition, anemia of the inner organs, chronic pulmonary emphysema, cardiac hypertrophy (650 grams), unsuspicious artificial aortic valves, medium-grade coronary sclerosis, and zones of scarred myocardium including fresh necrosis in the left ventricle were present.

Histology

Patient 1

Serial Sections of the Gallbladder Bed. These demonstrated marked vacuolization, decomposition, and intense shrinkage of the liver parenchyma with development of nonnucleated cell necrosis, zones of palisade-like alignment of cell nuclei, siderotic necrosis, blood congestion, and focal tiny bleeding in the adjacent liver tissue **(Figure 2)**. The adherent blood was partly responsible for a clear granulocytic reaction.

The liver had intense siderosis of stellate cells, little siderosis of the parenchyma, and focal necrosis of the parenchyma. Intense siderosis of the red pulp was found in the spleen. The pancreas had interstitial fibrosis and lipomatosis and no siderosis. Vasogenic atrophy and diffuse and nodular diabetic glomerulosclerosis were found in the kidney. The heart had little perivascular and interstitial fibrosis.

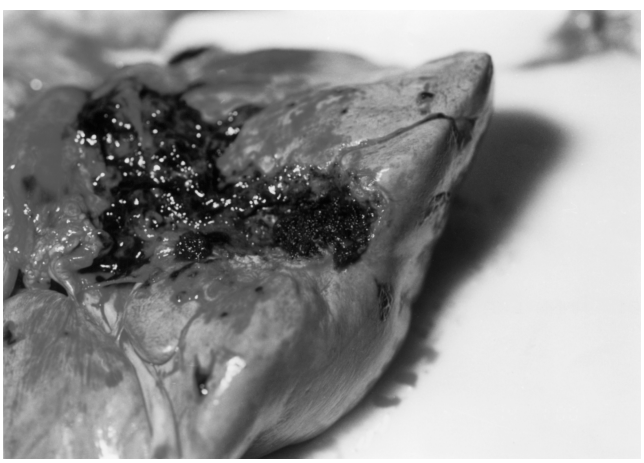

Figure 1. Liver (case 2): View of the gallbladder bed of the liver. Hemorrhage at the surgical site and hepatic porta. Evident fatty liver degeneration.

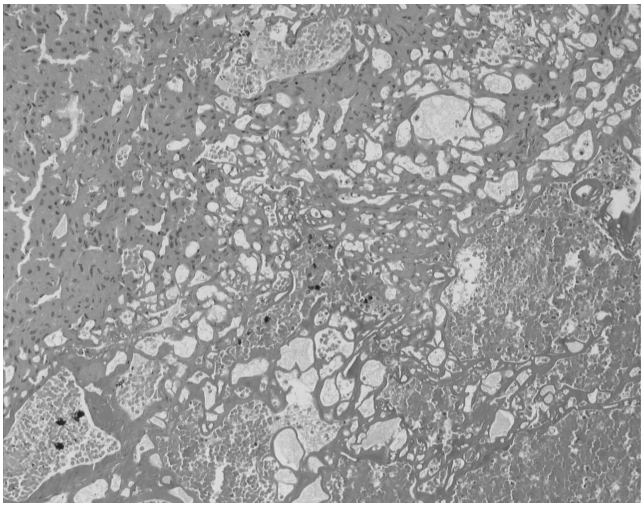

Figure 2. Liver (case 1): Vacuolic decomposition of the peripheral liver parenchyma (center of the figure) with transition to nonnucleated cell necrosis, which is a sign of thermal injury. Blood in the gallbladder bed of the liver (on the bottom right). (HE, original magnification 25x).

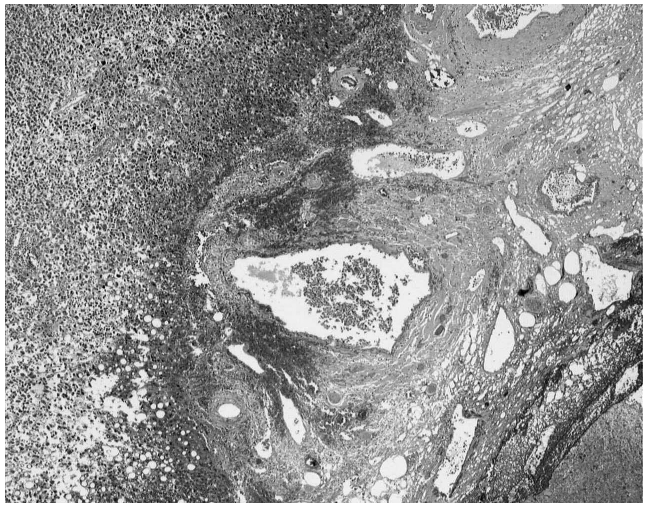

Figure 3. Liver (case 2): Callous, thickened gallbladder bed. Signs of thermal injury with vacuolic decomposition of the connective tissue of the gallbladder bed of the liver (right) and blood imbibed vascular walls (center) with increased perfusion. Blood with fibrin coats the liver surface (bottom right). Inhomogenous fatty degeneration of the liver. (Masson-Goldner, original magnification 10x).

Patient 2

Serial sections of the gallbladder bed. The gallbladder bed was callous, and the local fatty tissue was infiltrated by granulocytes. Some fresh blood, parenchymal necrosis, and some granulocytic reaction were below the liver capsule. Focal vacuolization and decomposition was seen in the connective tissue of the liver and the liver parenchyma, in part with palisade-like alignment of the cell nuclei. Further findings were focal blood in the vascular walls and blood coated to the liver surface of the gallbladder bed with moderate fibrin-rich zones **(Figure 3)**. Liver cells were bloated with fat to a higher grade, predominantly centroacinously. The periportal triangles were markedly infiltrated with lymphocytes. The heart muscle fibers were hypertrophic. Zones of old infarction aside fresh (some hours) necrosis of muscle fibres were obvious.

DISCUSSION

In both cases, the prosecution ordered preliminary proceedings to investigate the reasons for blood oozing. The medical experts disclosed the findings presented here. The cases were evaluated on the basis of the external hospital's charts and the autopsy conducted by the Institute of Legal Medicine, University of Hamburg. According to these data, the death of the first patient was caused by bleeding into the abdominal cavity and hemorrhagic shock. The blood in the abdominal cavity of the second patient was not enough to cause circulatory shock. However, the blood loss caused an extracardiac workload and increased the consumption of energy and oxygen. This put a great strain on the prediseased heart and caused a myocardial infarction. Therefore, bleeding was a factor that caused death.

The clinical courses and the autopsies in both cases revealed blood oozing from the gallbladder bed. Anastomotic branches of the cystic artery to the left or right hepatic artery were reported in 12%.[10] However, none of those vascular anastomoses were found in either case. Cystic arteries that lead from the right hepatic artery, run through the most cranial part of Calot's triangle, ascend between the gallbladder and the liver, and encircle the gallbladder with 5 to 7 branches have been identified in 2% of patients.[10] If the trunk of such arteries has not been ligated or identified, the surgeon has to deal with several small branches that have to be closed individually. This involves the risk of missing one or several small branches, which can result in persistent blood oozing. However, the autopsies of the 2 cases presented reveal correct closures of the cystic arteries. Injured blood vessels are another possibility for hemorrhage. However, this was also ruled out by autopsy.

Liver cirrhosis, consumptive coagulopathy, sepsis, and infection can cause diffuse seeping hemorrhage associ-

ated with operative procedures, but those findings were not true for these patients. However, the first patient was on hemodialysis due to renal insufficiency. Hemodialysis might be associated with the genesis of siderosis, and this was confirmed in the parenchyma of the liver and spleen of the first patient. In turn, siderosis can cause liver cell necrosis, which was histologically evident.

A series of postmortem experiments quantified the heat damage of the liver caused by cautery and laser coagulation.[11] Two effects were recognized. One was the direct thermal effect and caused cell necrosis. The other was transmission of heat resulting in swelling of parenchyma, damaged cell nuclei, and peliosis-like changes in the liver sinus with loss of the endothelium. In both cases, such histological changes resembling peliosis hepatis were obvious.

The inflammatory reaction of acute and subacute cholecystitis can involve the connective tissue next to the gallbladder bed and cause marked vascularization.[12] Those inflammatory alterations as well as fibrosis following chronic or recurrent cholecystitis increase perioperative morbidity.[13,14] Hence, they might be a relative contraindication for laparoscopic procedures and might increase the rate of conversion to conventional cholecystectomy. In the second case, such a constellation was obvious. To stop hemorrhage from the liver by thermal means could be difficult. In select cases, fibrin glue was recommended to stop diffuse blood oozing from the gallbladder bed.[12]

In both cases reported herein, the histological examination of the gallbladder bed of the liver revealed alterations increasing the vulnerability of the liver. In the first case, this was true for the siderosis, which was most probably also the reason for the necrosis of the liver parenchyma. In the second case, it was true for the fatty degeneration of the liver.

In summary, the exact cause of the blood oozing was undeterminable. Each of the mentioned factors (fatty liver degeneration, siderosis, alteration caused by chronic and recurrent cholecystitis, thermal injury to the liver parenchyma) was involved in initiating and perpetuating blood oozing. In the first case, hemorrhage was the sole cause of death. In the second case on the other hand, bleeding and existing heart disease led to death. Any quantification of the factors remains speculative.

According to the circumstances of death and autopsy findings, the fatal bleeding derived from the gallbladder bed. The bleeding must have derived from small vessels not detectable by gross or microscopic inspection. This assumption is supported by the results of the histological examination.

The medical experts came to the conclusion that both surgical procedures were *lege artis*, but the postoperative monitoring was criticized as fragmentary. The experts did not come to the conclusion that the death could have been prevented with forensic certainty even if the required postoperative standards had been adhered to. The charges in both cases were dropped.

In our point of view, it is not possible to make any recommendations concerning the frequency and the risk factors. These were the only cases in the last 10 years, and we therefore cannot offer any reliable recommendations.

An exact preoperative clinical and laboratory examination is essential to decide the correct surgical procedure (laparoscopic versus conventional surgery). Prevention of bleeding comprises the preoperative detection of risk factors that enable adequate preparation. Thorough surgical dissection in anatomical layers, the use of thermocoagulation, sutures, and clips can control bleeding. Local procoagulant drugs (eg, fibrin glue) are not routinely used but might be an option. Patients with an increased risk for complications after laparoscopic cholecystectomy should be monitored closely. Postoperative ultrasound examinations can detect fluid collections in the abdominal cavity, and a test for hemoglobin can detect its decline. Therefore, both measures can indicate hemorrhage and can be used as a rationale for an adequate action.

In case of lethal postoperative hemorrhage, the postmortem angiography can disclose an occult bleeding source.[15] A selective histological examination of the gallbladder bed should be mandatory for medical evidence.

CONCLUSION

Fatty liver degeneration, siderosis, chronic as well as recurrent cholecystitis, and thermal injury are able to initiate and perpetuate lethal blood oozing. Death can be caused either directly by exsanguination or indirectly by deteriorating preexisting morbidity. Histological examinations can disclose unknown risk factors of seeping hemorrhage.

References:

1. Muehe E. Die erste cholecystektomie durch das laparoskop. *Langenbecks Arch Chir*. 1986;369:804.

2. Bingener-Casey J, Richards ML, Strodel WE, Schwesinger WH, Sirinek KR. Reasons for conversion from laparoscopic to open cholecystectomy: a 10-year review. *J Gastrointest Surg*. 2002;6:800–805.

3. Shamiyeh A, Wayand W. Laparoscopic cholecystectomy: early and late complications and their treatment. *Langenbecks Arch Surg*. 2004;389:164–171.

4. Shea JA, Healey MJ, Berlin JA, et al. Mortality and complications associated with laparoscopic cholecystectomy. A meta-analysis. *Ann Surg*. 1996;224:609–620.

5. Frilling A, Li J, Weber F, et al. Major bile duct injuries after laparoscopic cholecystectomy: a tertiary center experience. *J Gastrointest Surg*. 2004;8:679–685.

6. Lai PB, Kwong KH, Leung KL, et al. Randomized trial of early versus delayed laparoscopic cholecystectomy for acute cholecystitis. *Br J Surg*. 1998;85:764–767.

7. Lujan JA, Parrilla P, Robles R, Marin P, Torralba JA, Garcia-Ayllon J. Laparoscopic cholecystectomy vs open cholecystectomy in the treatment of acute cholecystitis: a prospective study. *Arch Surg*. 1998;133:173–175.

8. Puggioni A, Wong LL. A metaanalysis of laparoscopic cholecystectomy in patients with cirrhosis. *J Am Coll Surg*. 2003;197: 921–926.

9. Lee VS, Chari RS, Cucchiaro G, Meyers WC. Complications of laparoscopic cholecystectomy. *Am J Surg*. 1993;165:527–532.

10. Bergamaschi R, Ignjatovic D. Anatomic rationale for arterial bleeding from the liver bed during and/or after laparoscopic cholecystectomy: a postmortem study. *Surg Laparosc Endosc Percutan Tech*. 1999;9:267–270.

11. Hoffmann R, Flury R. Laparoscopic cholecystectomy: heat damage to the liver caused by hooked electrode and laser. *Helv Chir Acta*. 1994;60:961–964.

12. Kockerling F, Schneider C, Reymond MA, Hohenberger W. Controlling complications in laparoscopic cholecystectomy: diffuse parenchyma hemorrhage in the liver parenchyma. *Zentralbl Chir*. 1997;122:405–408.

13. Giger U, Michel JM, Vonlanthen R, Becker K, Kocher T, Krahenbuhl L. Laparoscopic cholecystectomy in acute cholecystitis: indication, technique, risk and outcome. *Langenbecks Arch Surg*. 2004;DOI:10.1007/s00423–004-0509–4.

14. Michalowski K, Bornman PC, Krige JE, Gallagher PJ, Terblanche J. Laparoscopic subtotal cholecystectomy in patients with complicated acute cholecystitis or fibrosis. *Br J Surg*. 1998; 85:904–906.

15. Lasczkowski G, Riepert T, Mattern R. Letale spätblutung bei cholecystektomie - Aufdeckung okkulter blutungsquellen mittels postmortaler angiographie. *Rechtsmedizin*. 1992;2:83–86.